A Paradoxical Diurnal Movement Pattern in Obese Subjects With Type 2 Diabetes

A contributor to impaired appetite and glycemic control?

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Observational studies have found an association between self-reported impaired sleep quality and subsequent risk of type 2 diabetes (1,2). In a German cohort of 4,140 men and 4,129 women (aged 25–74 years), sleeping difficulties were associated with higher risk of developing type 2 diabetes during the mean follow-up period of 7.5 years (1). In the Nurses Health Study (70,026 U.S. women aged 40–65), short sleep duration (≤5 h per day) was associated with risk of incidents of diabetes over a 10-year period (2). A few short-term trials have demonstrated that sleep restriction is associated with increased sympathoadrenal secretion of cortisol and catecholamines (3,4), decreased evening leptin, and increased ghrelin levels (5). Chronic increased sympathoadrenal activity may alter glucose homeostasis and induce insulin resistance. Thus, impaired sleep quality may act as a stress factor, stimulating appetite, promoting weight gain, and impairing glycemic control, with subsequent increased risk of type 2 diabetes.

The weakness of previous studies is the self-reporting of sleep quality and risk of confounding by associated traits. We measured movement during sleep in type 2 diabetic patients and control subjects by an objective technique and compared the sleep problems of obese subjects with type 2 diabetes with those of nondiabetic obese subjects.

RESEARCH DESIGN AND METHODS — Data from 24-h whole-body calorimetry measurements were used. All subjects in the type 2 diabetic group ($n = 31$) were diagnosed according to the American Diabetes Association criteria from 1997 (6). The control group was compiled from a database and consisted of 61 nondiabetic and healthy subjects with similar body composition as the type 2 diabetic subjects. Details on data collection and study protocols are described elsewhere (7).

Twenty-four–hour energy expenditure was measured continuously by indirect whole-body calorimetry (8). Spontaneous physical activity level (SPA) was measured by two microwave radar detectors (Sisor Mini-Radar; Static Input System, Lausanne, Switzerland) continuously emitting and receiving a signal. The SPA measurements indicate the percentage of time the subjects were active to a detectable degree. Heart rate was continuously monitored by a telemetry system (Dialogue 2000; Danica Electronics, Copenhagen, Denmark). Twenty-four–hour energy expenditure, heart rate, and SPA were divided into two time intervals for analysis: daytime (9:00 A.M.–11:00 P.M.) and sleep (1:00 A.M.–6:00 A.M.).

Variables that were not normally distributed were either log transformed or categorized as appropriate. General linear modeling was used to compare SPA and energy expenditure between the groups after adjustment for body composition, sex, and age. Statistical analyses were performed with SPSS for Windows, version 11.0.1 (SPSS, Chicago, IL).

RESULTS — A total of 31 obese subjects with type 2 diabetes and 61 nondiabetic obese control subjects were analyzed (61% women in both groups). Only age differed significantly between the two groups (59 ± 9 vs. 43 ± 6 years, $P < 0.001$). No significant differences in body weight and composition were observed. The mean BMI (± SD) was 34.7 ± 4.3 kg/m^2, and fat-free mass was 39.6 ± 7.3% (7).

In univariate analyses, age was negatively associated with daytime SPA and positively associated with sleeping SPA ($P = 0.034$ and $P = 0.051$, respectively). Common hemodynamic side effects related to type 2 diabetes (e.g., hypertension, dyslipidemia, and headache) were not associated with either daytime or sleeping SPA. Therefore, only age was included as a covariate in all analyses.

Mean (±SE) sleeping SPA was 59% higher in the type 2 diabetic group than in the nondiabetic group (0.92 ± 0.13 vs. 0.58 ± 0.08%, $P = 0.041$) analyzed with age as covariate. Basal metabolic rate was 7.6% higher (+145 ± 20 kcal/day) than sleeping energy expenditure in the control group, whereas it was only 3.6% (+72 ± 27 kcal/day) higher in the type 2 diabetic group ($P = 0.031$). In contrast, mean daytime SPA was significantly lower (−11%) in the type 2 diabetic subjects than in the nondiabetic subjects (11.4 ± 0.5 vs. 12.7 ± 0.3%, $P = 0.045$). No significant differences between the type 2 diabetes and control groups were found in daytime heart rates (81.9 ± 1.4 vs. 83.7 ± 2.2, $P = 0.554$) or sleep heart rates (66.6 ± 1.4 vs. 69.9 ± 2.2, $P = 0.276$) before or after adjustment for age (Table 1).

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Abbreviations: SPA, spontaneous physical activity level.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

Table 1—*Whole-body indirect calorimetry*

	Type 2 diabetes	No diabetes	Mean difference
n	31	61	
Daytime SPA (%)†	11.3 ± 0.5	12.8 ± 0.3	−1.5 ± 0.7 (−12%)‖
Sleeping SPA (%)†	0.92 ± 0.13	0.58 ± 0.08	+0.34 ± 0.2 (+59%)‖
24-h energy expenditure (kcal/day)	2,587 ± 61	2,563 ± 43	+12 ± 74 (+0.9%)‡
Adjusted 24-h energy expenditure (kcal/day)*	2,679 ± 37	2,515 ± 23	+164 ± 31 (+6.5%)§
Basal metabolic rate (kcal/day)	2,060 ± 59	2,042 ± 42	+18 ± 73 (+0.9%)‡
Adjusted basal metabolic rate (kcal/day)*	2,140 ± 45	2,001 ± 28	+139 ± 61 (+6.9%)‖
Daytime energy expenditure (kcal/day)	2,941 ± 72	2,995 ± 51	−54 ± 89 (−1.8%)‡
Adjusted daytime energy expenditure (kcal/day)*	3,036 ± 46	2,948 ± 29	+89 ± 64 (+3.0%)‡
Sleeping energy expenditure (kcal/day)	1,989 ± 51	1,897 ± 37	+92 ± 63 (+4.8%)‡
Adjusted sleeping energy expenditure (kcal/day)*	2,023 ± 29	1,879 ± 18	+144 ± 40 (+7.6%)§
Daytime heart rate (bpm)†	83.5 ± 2.3	82.0 ± 1.4	+1.5 ± 3.1(+1.8%)‡
Sleeping heart rate (bpm)†	70.2 ± 2.2	66.5 ± 1.4	+3.7 ± 3.1(+5.6%)‡

Data are means ± SE. *Adjusted for fat-free mass, fat mass, SPA, sex, and age; †adjusted for age. No significant difference between groups: ‡$P > 0.05$; significant difference between groups: §$P < 0.01$; ‖$P < 0.05$.

CONCLUSIONS — The type 2 diabetic subjects were more sedentary in daytime than nondiabetic control subjects. Our results suggest that the type 2 diabetic subjects have impaired sleep quality compared with the control group. This may be due to difficulties falling asleep or restlessness from snoring, apnea, thirst, or urination. Our results are in agreement with other studies suggesting that impaired sleep quality and duration may be closely related to glycemic control and insulin resistance (3,4).

An obvious consequence of impaired sleep quality may be indisposition and tiredness, leading to less activity during the day. In the long term, sedentary habits may cause weight gain and deterioration of the diabetic state. It is well established that low levels of physical activity are a strong risk factor for type 2 diabetes (9) and that sedentary behavior increases with increasing body weight.

It is notable that the nondiabetic subjects were younger than the type 2 diabetic subjects. Age was correlated with the level of both sleeping and daytime SPA, indicating that advanced age may have a negative effect on sleep quality and activity level during the day. The first is in agreement with a large cohort study in which elderly adults reported difficulties falling asleep and maintaining sleep (10). Age was therefore included as a covariate in all analyses. The group difference remained after age adjustment.

Taken together, the paradoxical diurnal movement pattern in type 2 diabetes suggests impaired sleep quality and duration, which may negatively affect glycemic control and weight regulation. We have previously reported findings of a 7% higher basal metabolic rate and 24-h energy expenditure under standardized low level of physical activity, which remained after adjustment for fat-free mass, fat mass, SPA, sex, and age in the type 2 diabetic subjects compared with nondiabetic subjects (7). In theory, the higher energy expenditure would be expected to facilitate a negative energy balance and therefore weight loss in obese type 2 diabetic patients. However, it is well established that type 2 diabetic patients are often more resistant than matched nondiabetic individuals to losing weight on weight management programs (11). Therefore, the paradoxical diurnal movement pattern and disrupted appetite regulation may be more important than the higher energy expenditure under free-living conditions.

Further longitudinal studies on the metabolic and physiologic changes associated with the development of type 2 diabetes are warranted.

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